# Letter to the Editor "Vibroacoustic Disease" and Response

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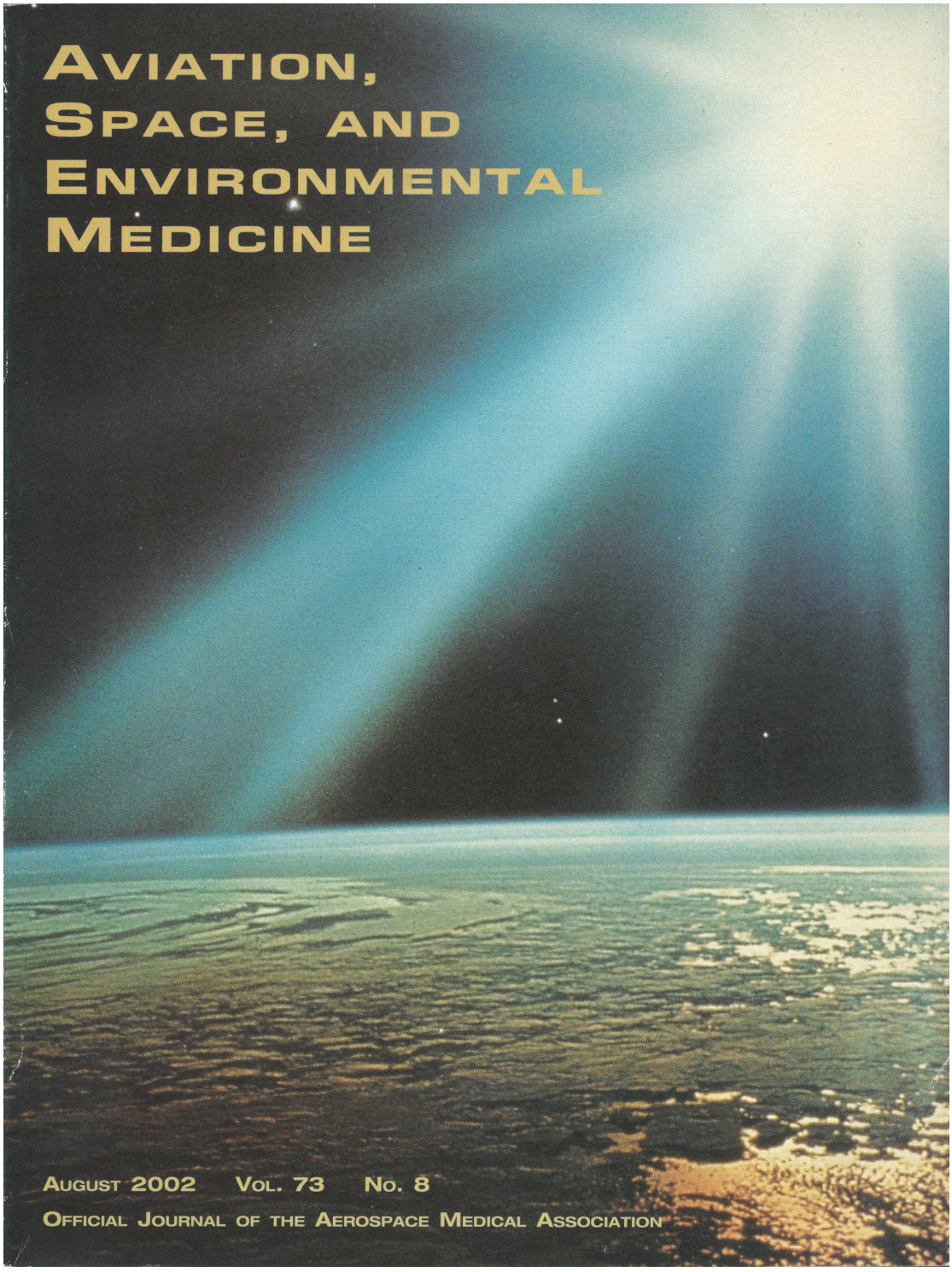


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## "Vibroacoustic Disease"

Dear Madam:

"Vibroacoustic disease" was introduced to the medical literature as a new "emerging pathology" with the Supplement to this journal in 1999 (Aviat Space Environ Med 1999; 70 (3 Section II). The contents and conclusions of this 154 page supplement with over 20 studies did not undergo full peer review and can hardly be accepted as an authoritative contribution to the scientific literature until they are published in an authoritative, peer-reviewed form. However, since the findings are now available and might be quoted, they certainly deserve discussion and scientific criticism.

Past studies on the subject matter are covered in the scientific literature under "non-auditory effects of noise" and have so far failed to provide the clear cut criteria and dose-response relationships we would like to have to control occupational and public health exposures in industry, community settings, and military environments. However, in spite of the complexity and elusiveness of the subject, there have been many attempts to study these effects since the classical Benox report in 1953 (1) called attention to the magnitude of this problem. The supplement hardly mentions the surveys, experiments, programs, regulations, and recommendations published by the U.S. Environmental Protection Administration, National Institute of Occupational Safety and Health, Occupational Safety and Health Administration, and Department of Defense, nor does it mention the hundreds of published reports on all operational U.S. aircraft noise environments and noise levels on maintenance positions and test stands (2). Most of these data are more detailed and useful for personnel protection than the surveys on the identical aircrafts included in the Supplement. The National Academy of Sciences—National Research Council reports on noise effects, vibration effects, and protection measures are not discussed. The relationships and implications of the findings for the various noise, impulse-noise, and vibration standards of individual countries and of the International Standards Organization (ISO) and the recommendations of the World Health Organization (WHO) on noise (10) as well as vibration are not touched on. Awareness of this body of information should have restricted the reported findings to the commonly used classification "non-auditory health effects of noise," since the whole supplement presents no evidence of structure-born vibration transmitted to the human body. There is no need to define a new disease.

There appears to be little doubt that, based on all previously published data (3), there is sufficient evidence for a causal relationship between noise exposure and some non-auditory health effects (8). Primary attention has been focused on the potential noise effects on hypertension and ischemic heart disease. Most experts agree that adherence to the occupational hearing conservation criteria and environmental outdoor and indoor exposure goals reduces the risk of non-auditory health effects to insignificant levels (7,8). Populations exposed occupationally to potentially hazardous situations should be monitored by a hearing conservation program, for which detailed international guidelines exist (ISO, WHO) and which consists of three elements: 1) education concerning the noise hazards; 2) education in the proper use and supervision of the wearing of hearing protection; and 3) monitoring audiometry, including periodic medical examinations. In the regulations for the U.S. and most other countries, the noise hazard starts at 85 dB(A) and protection becomes mandatory at 90 dB(A) over an 8-hour workday. Exposure to higher doses is illegal and certainly unprofessional and irresponsible.

All these guidelines, regulations and criteria have existed for over 25 years. Irrespective of this, the coordinators of the subject supplement present data of their studies supposedly documenting evidence for this "new pathological entity" caused by long-term (≥10 yr) exposure to sound pressure amplitudes over 90 dB(A). The obvious questions one has to ask and which are not answered by the supplement are:

1. What were the individually measured or estimated sound levels, $L_{Aeq}$ for the daily, weekly, and yearly exposures of the subjects? What were the exposure doses for the postulated three clinical stages? (Approximately 5, 10, and over 10 exposure years.)
2. What were the age- and noise-induced hearing losses of the patients? The noise-induced permanent threshold shifts after 10 yr of exposure for 8 h per workday to 100 dB(A) should have been over 20 dB for 10% of the population averaged across audiometric frequencies (ISO standard 1999). This added to the age-associated hearing loss, must have resulted in an appreciable hearing handicap (4). No audiograms or statistical data on their changes with time are presented.
3. The well-known psycho-social effects of such hearing losses are not considered in several of the papers.
4. To what extent was hearing protection used and enforced? What protectors? For the frequency range postulated as particularly dangerous for "vibroacoustics diseases" (VAD) (≤500 Hz), ear protection of up to 20 to 40 dB is available for most of the range (125–500 Hz). Statements (page A16) such as "Workers who did not use their hearing protection devices. . ." indicate that enforcement was absent.
5. What support can be offered for the statement on page A16 that the "researchers have long been aware that hearing protection, in reality, promoted the development of VAD," because workers, who did not use hearing protectors, walked away from the noise to allow for some recovery?
6. Why do the studies in the supplement on cardiovascular effects and potential airway-lung injury neglect recent studies, which try to establish a relationship between noise exposure dose and effect incidence (5–8)? Particularly, studies on non-auditory injury criteria for repeated blast overpressure exposures and computer modeling of thoracic response appear relevant (5).

These are just some of the most important questions. Without satisfactory response to these questions, any further serious discussion and criticism of the individual clinical reports is superfluous. Hopefully, with strict adherence to a hearing conservation program and personal noise dosimetry—well within the state-of-the-art for decades and successfully applied to many industrial noise situations and to the general population at large (9)—many of the reported findings might disappear.

There is no question that non-auditory health effects from noise exposure exist (8), particularly in small sensitive subgroups of the population. We certainly support scientific studies of the problem, particularly over years of documented exposure. New findings might emerge from statistical studies of the large number of data banks already available following enforcement of hearing conservation programs in industry and the military services of many countries. However, publication of results as a supplement lacking rigorous peer review could lead to unjustified compensation claims and litigations. They could also lead to medical malpractice by not insisting on the best ear protection available. "Vibroacoustic disease" remains an unproven theory belonging to a small group of authors and has not found acceptance in the medical literature.

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In Response:

In response to the letter authored by Dr. H. Von Gierke and Dr. S. R. Mohler, we have the following commentary.

Low frequency noise (LFN)-induced pathology was introduced to the medical literature in the early 1980s (9–11). In 1984, the Portuguese National Public Health Award was presented for our work, which was published the following year in the Journal of the Medical Sciences Society of Lisbon (2,3). This topic was introduced to the English medical literature, via the Aerospace Medical Association (AsMA), in the early 1990s. In 1995, the European Commission of Health and Safety took notice of our work (8) and all data collected to date was compiled in the March 1999 Supplement to this journal (Vol. 70, No. 3, Section II). In 2001, our work on mutagenic effects of LFN and vibration exposure, in human and animal models, received the Portuguese National Public Health Award (16). As for the lack of a full peer-review of the work compiled in the Supplement, we believe that the pre- and post-publications (some referenced here), speak for themselves.

Non-auditory effects of noise have been inconclusively studied because: a) the noise in question is inadequately assessed; and b) the clinical evaluations are either subjective (i.e., annoyance) or limited to standard evaluations (i.e., hearing loss, hypertension, sleeplessness, etc.). How can the effects be ascertained if the agent of disease is not being appropriately quantified *and* the associated clinical manifestations are being overlooked? This is why "the Supplement hardly mentions the surveys, experiments, programs, regulations, and recommendations . . . ." Since we investigated the effects of *low frequency* noise, and the vast majority of these surveys, experiments, and "hundreds of reports" **do not** provide a frequency spectrum analysis, they were deemed non-applicable to our discussion. Measuring noise merely in terms of dB (or dBA) gives no information on the frequency distribution of the acoustic energy. It is not scientifically valid to compare two noise-related studies when one describes the acoustic environment merely in terms of the overall amplitude (in dB or dBA), while the other provides a frequency distribution analysis of acoustic energy.

"Most experts agree that adherence to the occupational hearing conservation criteria . . . reduce the risk of non-auditory health effects to insignificant levels." We must clarify concepts. If *non-auditory effects* are, by definition, effects outside of the auditory system, then their relationship with the use of hearing protection devices should be a moot point. However, if the authors are referring to effects that are non-auditory, but whose pathway is *via the auditory system*, then, clearly, it is incoherent to call them non-auditory effects. We believe that we have demonstrated that much of vibroacoustic disease (VAD) related pathology is **not** induced via the auditory system. We postulated that airborne acoustic energy, impacting on viscoelastic tissues, could be responsible for much of the observed pathology. Usually, VAD patients do not suffer the known consequences of solid-to-solid vibration, i.e. structure-born vibration.

The authors' insistence in describing international guidelines related to noise exposure suggests that they are unaware that LFN is not recognized as a health hazard. "Education concerning the noise hazards" regarding LFN does not exist. "Monitoring audiometry, including periodic medical examinations" will *only* focus on professional hearing loss at the 4000 Hz notch. When significant losses only in the 250–500 Hz bands are detected—a clear indication of LFN exposure—no action is required to protect the worker. The fact that "all these guidelines, regulations, and criteria have existed for over 25 years" should be an irrelevant factor in the pursuit of scientific truth, should it not? Or, should the non-existence of asbestos legislation have hindered the inquiry into its possible deleterious effects?

Responding to the authors' direct questions:

1. On pp. A24–5 of the Supplement, the graphs representing the frequency distribution analysis of aircraft noise show the average amplitude of the acoustic environment, in dB(A) and dB(Lin). In Fig. 6 (or Fig. 10), where the acoustic energy is concentrated in the lower frequency bands, notice the difference between the dB(A) and dB(Lin) levels. Clearly, if the acoustic energy is concentrated in the lower frequency bands, dB(A) measurements are misleading, to say the least. Thus, $L_{A_{eq}}$ values would be quite useless.

   Dose-response relationships are among the most difficult parameters to determine, especially where ubiquitous environmental agents are concerned. In "Clinical Stages of VAD" (p. A32) we described the time it took for 50% of a rigorously selected (p. A2) study population to develop the corresponding sign or symptom. This is an approximate measure of a dose-response, and similar animal data can be consulted in the Supplement. LFN also exists outside the workplace, and this presents a substantial difficulty in determining dose responses. In fact, LFN is ubiquitous in much of our daily lives, but to obtain good dose response measures, non-LFN-exposed individuals are required. So who would be good controls for VAD-related studies? False controls (p. A148) in VAD-related studies are frequent because standard selection criteria are simply **not** applicable. A specific example: the study conducted by scientists from the Ponce School of Medicine (PSM), Puerto Rico, on the existence of cardiac pathology among the residents of the Island of Vieques. Evaluated by cardiologists from the Mayo Clinic, the PSM report was determined to be junk science (1). However, our study of the same population (17) unmistakably demonstrated a highly statistically significant difference between control and diseased populations. The problem with the PSM study, the report of which was sent to us for evaluation, was the inadequate selection of a control population. By definition, VAD control populations *cannot* be exposed to LFN. This condition was not guaranteed in the PSM study. In human models, dose responses to an agent of disease that is ubiquitous, whose effects evolve over long periods of time (measured in years), and which is inadequately measured during standard assessment procedures, are quite a challenge for any team.
2. Age- and noise-induced hearing losses of VAD patients were first presented in English at the 1996 AsMA Annual Meeting in Atlanta (15). However, in Portugal, publications regarding the hearing of LFN-exposed personnel began in the early 1980s (2,3,9,11). As discussed previously, in 134 VAD patients (see selection criteria, p. A2), only 5 had hearing losses in the order of 50 dB at the 4000 Hz notch. Fifty-seven percent had losses $> 30$ dB, but at 250–500 Hz. All had thickening of some cardiac structure (15).
3. The "well-known psycho-social effects of such hearing losses" are not considered because this is not pathology associated with LFN exposure. On the contrary, the psychosocial effects of LFN exposure (i.e., hypersensitivity to noise, increased aggressiveness) were previously investigated (9) and included in the Supplement (p. A145).
4. In 1980, when these studies began, hearing conservation programs had only begun to be implemented a few years earlier. Therefore, many of the older workers still exhibited the "macho" pride of not requiring such "sissy devices" for protection. After all, they had been working without protectors for so long. Certainly this attitude was encountered in industries all over the world. Enforcement came later, and today hearing losses among these workers have substantially decreased.

   Since noise protection is solely equated with hearing conservation, appropriately all noise protectors are hearing protection devices. However, ear protection, while magnificent for the pro-

tection of the auditory system, does nothing for the whole-body impact of LFN. The best analogy is that of light. X-rays and visible light are the same electromagnetic phenomena, but at different frequencies. The human eye does not perceive X-rays, but it does perceive visible light. For protection against the visible light, dark glasses are used. In the same way that dark glasses are inappropriate to protect against X-rays, ear protectors are inappropriate to protect against LFN.

5. In the individuals who avoided wearing hearing protectors, VAD pathology was observed to be more severe than in their colleagues who did use their protectors. This was an *observation* that was clinically evident, and corroborated both by the individuals and their colleagues. Those who did not wear their protectors made more frequent breaks from work—either by going for a smoke, a coffee, or to chat with co-workers. This, too, was corroborated both by the individuals and their colleagues. If the concepts underlying the human auditory system and the physics of acoustic phenomena are well understood, this observation is not surprising. By not using ear protectors, the individual removes himself from the LFN environment. It is the frequent removals from the environment that attenuate the severity of the pathology—not the non-use of ear protectors (!). The individual removes himself from the environment because he feels bothered by the *auditory noise.* The organ of alarm—the ear—serves (one of) its purposes. However, if such organ of alarm is cancelled by ear protectors (which will, however, correctly protect the individual's hearing), certainly the individual will permit himself to remain in such hostile environment, thus being overall exposed to LFN for longer periods of time. Moreover, an interesting study on the symptomatic complaints of boiler plant workers before and after the implementation of a hearing conservation program, showed no decrease in the amount of symptomatic complaints (7).
6. Past studies that tried to establish a relationship between noise and exposure dose effect incidence do not, in general, include a frequency distribution analysis. On the other hand, the effects studied are often limited to hearing losses, hypertension, annoyance, etc., which, again, are not comparable with our studies. Blast overpressure exposures were touched upon as airborne acoustic pressure waves that could impact the organism in much the same way as LFN (p. A17). Of course, the amplitude and time span over which both events occur is entirely distinct, but the effects are not (p. A17).

We have focused much of our time on the respiratory effects of LFN exposure. In 1996, a member of our team was awarded the AsMA Ross McFarland Young Investigator Award for determining that LFN exposure interferes with the normal immune response of rat pleura. In 2000, our work was awarded first prize by the Portuguese Society of Cellular Biology and Electron Microscopy; in 2001, the National Award for Occupational Health, and our most recent publications on LFN-induced respiratory pathology continue to support the concept that LFN targets the respiratory system (5,14). Dr. Von Gierke himself co-authored two articles in the 1960s (6,13) that unmistakably showed LFN having a deleterious effect on the respiratory system of volunteers which included coughing and gagging sensations (among others) after an exposure of mere minutes (p. A15). Surely it is not speculated that the use of ear protectors here would have prevented these subjective responses.

The authors' opinion that there is no need to define a new disease is very unfortunate. Thickening of the pericardium in the absence of an inflammatory process and with no diastolic dysfunction is the hallmark of vibroacoustic disease (12). There is a clearly defined agent of disease (which still goes unchecked everyday), specific symptoms that subside or disappear after periods of time away from LFN environments, and prevention and monitoring programs that have yielded successful and validating results (p. A148).

"Hopefully, with strict adherence to a hearing conservation program . . . many of the reported findings might disappear." We would very much like to share this optimism, but scientific reality grounds us to the obvious: until the lower frequency components of acoustic environments are recognized as existent, and are reliably measured, we do not foresee a disappearance of these findings (4).

The authors' fear that our *non-peer-reviewed* work published in the Supplement might be the basis of "unjustified compensation claims and litigations" strongly suggests that this may be the true motivation for their letter, but beyond the scope of our scientific investigations. The idea that any medical doctor, worthy of the Hippocratic Oath, would even *consider* suggesting the non-usage of ear protectors to avoid VAD is, in our view, absolutely absurd.

We invite the authors to study the acoustic environments of the personnel under their care, and perform echocardiograms on these individuals. Publish the results. Our data could then be *scientifically* confronted.

**Nuno A. A. Castelo Branco, M.D. and Colleagues**
**Head, Scientific Board**
**Center for Human Performance, Portugal**

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